We claim:

- 1. A compound which specifically alters the binding activity of SR-BI, in combination with a pharmaceutically acceptable carrier, in an effective amount to treat a human or animal in need thereof, obtained by screening a library of compounds for alteration of SR-B1 binding activity or expression.
 - 2. The compound of claim 1 selected from the group shown in Table I.
- 3. The compound of claim 1, selected from the group consisting of BLT-1 (MIT 9952-53), BLT-2 (MIT 9952-61), BLT-3 (MIT 9952-19), BLT-4 (MIT 9952-29), and BLT-5 (MIT 9952-6).
- 4. A method for altering cholesterol transport into or out of cells comprising inhibiting expression or activity of SR-BI comprising administering to an animal or human in need thereof the composition of claim 1.
- 5. The method of claim 4, wherein the composition of claim1 enhances HDL binding by increasing SR-BI's binding affinity for HDL.
- 6. The method of claim 4, wherein the inhibited SR-BI binding activity blocks SR-BI-mediated lipid transport.
- 7. The method of claim 6, wherein the inhibited SR-BI binding activity blocks SR-BI-mediated selective lipid uptake.
 - 8. The method of claim 7, wherein the lipid is HDL cholesteryl ether.
- 9. The method of claim 4, wherein the inhibited SR-BI binding activity blocks efflux of cellular cholesterol to HDL.
- 10. A method of identifying a compound which alters SR-BI binding activity or expression comprising screening a library of compounds.
- 11. The method of claim 10, wherein the SR-BI expression is determined by Northern analysis.
 - 12. The method of claim 10, wherein the library is a chemical library.
 - 13. The method of claim 10, wherein the SR-BI binding activity is inhibited.
- 14. The method of claim 13, wherein the inhibited SR-BI binding activity blocks SR-BI-mediated lipid transport.
- 15. The method of claim 14, wherein the inhibited SR-BI binding activity blocks SR-BI-mediated selective lipid uptake.

- 16. The method of claim 15, wherein the lipid is HDL cholesteryl ether.
- 17. The method of claim 10, wherein the inhibited SR-BI binding activity blocks efflux of cellular cholesterol to HDL.